# Lung Cancer Among Black And White Migrants In The U.S.*†

## Etiological Considerations

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Studies of disease distribution among different population groupings and aggregates have often furnished important clues for disease antecedents but because aggregate population data can deal at best only with gross characteristics, differences that occur in the disease incidence among different populations need to be dramatic and related to well established properties before any firm conclusions can be drawn from such data. This broad approach of descriptive epidemiology has focused on the major scientific unknowns: host response, susceptibility, resistance and adaptability, the influence of the environment and the interrelationships of the host with the environment.

An important observation in these studies has been the marked individual variations in susceptibility and resistance to carcinogenic agents. Among a group of workers exposed to an occupational carcinogenic agent, some develop the specific cancer and others do not. Host factors, inherited or acquired, exclusive of exposure considerations, is an area of research which has been virtually unexplored in industrial studies. The present investigation is concerned with the critical examination of the interaction between environmental and host factors.

Data obtained from migrant populations have generally supported the hypothesis that the complex environment may contribute the major carcinogenic potential in the etiology of lung cancer. In general, lung cancer mortality for migrant populations has been observed to fall between the rates in country of origin and the new host country. This observation has been established predominantly for English immigrants to Australia, Canada, New Zealand, South Africa and the United States. It has been observed for Jewish immigrants to Israel, the United States and Canada, and for Italian and Mexican immigrants to the United States.[1-10]

Not only does the change in mortality patterns for lung cancer point toward environmental factors, but in some instances the direction of the increase or decrease is related specifically to the difference in pollution between the country of birth and the country of migration. English migrants, who came from a highly polluted environment and moved to countries with lower levels of pollution, experienced a carryover but nevertheless a decrease in lung cancer mortality as did Jewish migrants from northeast Europe who moved to the less polluted environment of Canada or Israel.

However, these findings are at best suggestive since knowledge of a migrant's original environment is only approximate. Death certificate studies sometimes present tantalizing information without satisfying the inquisitive appetite.

It is possible that much more precise information about lung cancer may be obtained

*Presented at the 1973 Meeting of the South West Section of the American Association for Cancer Research, Houston, Texas – November 9 and 10, 1973

†Work supported by Research Career Development Award 1-K04-CA-18219-01, National Cancer Institute and by Special Foundation Fund, Council for Tobacco Research, U.S.A., Inc.

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from migration patterns within the United States. Sauer previously reported that in general for whites in the country as a whole, those who had moved to another state had a higher lung cancer death rate than did those born in their state of residence even though the all-causes death rates were quite similar for the two groups.[1] Haenszel found an increased risk for lung cancer among white males who migrated from rural to urban areas.[12] Mancuso, in comparative studies by place of birth within the United States and migration, demonstrated a nonwhite excess over the native-born white males for lung cancer and other cancer sites.[9]

A constant migration has taken place, especially of the black population, from the American South, the American Northeast and the Northwest.[4] A numerous population, largely rural in origin, migrated from the South to the industrial Northeast and North Central states, and to the West Coast from 1940 to 1970. The pattern of black migration rapidly accelerated with World War II when 1.6 million blacks, nearly one sixth of the total southern black population migrated north and west in search of jobs and new opportunities. The exodus from the South continued in a roughly comparable pace during the decade of the fifties and diminished only slightly during the sixties.

There has been a consistent rise in lung cancer mortality among blacks, in particular the males, which is compatible with the concept of migration for employment and the subsequent latent period for environmental effects. Starting with 1960 for males, the age adjusted cancer death rates for blacks passed that for whites and since then this disparity has steadily increased.[1] It must be recognized, however, that the increase in total cancer in the United States black population is not of racial origin. While it could be possible that the increase in cancer was an apparent result, due in part to improved medical services for the black population, the marked differences could not be explained on this basis.

The excess of lung cancer among the nonwhite males compared to white, has been previously noted. The more dramatic increase in lung cancer among the black males and the females was signalled by the reports of Burbank[13] and of Schneiderman and Levin.[14]

It is possible that a clue for the etiology of lung cancer may emerge from the comparison between the age adjusted mortality of migrants and of non-migrants, especially among blacks. Data from the State of Ohio were used for this comparison. Ohio has a large black population which was rapidly and dramatically increased by migration from the South. Not only did the new migrants come from an impoverished rural agricultural background but for a large part they found work initially as industrial laborers and subsequently as craftsmen and operators, work which exposed them to various concentrations of chemical dusts and fumes.

## METHODOLOGY

The population examined in detail consisted of Ohio residents born in the United States or in the large regions grouped by the census reports as the South. All deaths of Ohio residents, who died in the United States and Canada, were included in the survey. Population data, by specific characteristics used as a basis for computing death rates, are from the 1960 U.S. Census. In these census reports, nonwhites include Negroes, Indians, Chinese, Japanese and other "nonwhite" individuals. However, the nonwhites who were born in the U.S. and migrated from the South to Ohio or were born in Ohio are for the most part Negroes so that nonwhite and black may be used interchangeably for our study.[4] The place of birth for the deceased was taken from information provided for the death certificate by relatives or other persons completing the certificate. The region defined as "South" and used for classification of place of birth was based on Census groupings of the States. Age groupings were based on the age of the person on the last birthday. Average annual age specific death rates by color, sex and nativity were computed on the basis of population data in the 1960 U.S. Census for ages of 35 years and older. The populations for the intercensus years were estimated using a linear interpolation based on a comparison of a number of different methods for estimating populations

[4] ... Distribution of U.S. Census Population

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in intercensus years that yielded largely identical results).[15] Siegel's comprehensive analysis of the 1960 and 1970 Census data determined that the major portion of the under reporting of the nonwhites in 1960 occurred under the age of 35 (preferred estimates Table 5 Set D).[16] Death rates for the age span 35-69 years and 35+ were age adjusted by the direct method on the basis of the age distribution of the total population of continental United States in 1960. Data on specific dates of migration for population groups were not available for consideration.

## FINDINGS

The age specific death rates and age adjusted mortality rates per 100,000 for the years 1959-67 for Ohio residents by place of birth and race for males and females, were tabulated.*

There is no difference between the white and the black† males who were born in Ohio. The mortality experienced for migrants, however, is quite different. On the average, white migrants experienced a 50% increase and black migrants a 100% increase in age adjusted mortality. While there are some differences in the age specific rates between whites and nonwhites who were born in Ohio, the differences are nominal when compared to the differences between native Ohioans and migrants.

Similar differences, but less striking, occur among the females. White females born in Ohio have the lowest lung cancer rate followed by black females born in Ohio, followed by white females born in the South, followed by black females born in the South.

The findings show that the dramatic increase in lung cancer among black males is limited to migrants. Thus, the apparent increase in lung cancer for the total black population is due principally to that part of that population that migrated from a rural non-industrial to an urban-industrial environment.

## DISCUSSION

Migrant studies generally examine some association of genetic, environmental and economic factors endemic in particular areas and population subgroups, which in the early years of life provide social and biological imprints that combine with subsequent migration and environmental influences to bring about biological imbalances which supply the medium for the development of specific disease, disability and mortality risks.[9]

A number of specific hypotheses may be advanced and examined in this point.

Some authors in noting the increase of lung cancer among nonwhites raised the possibility that the increase may be related to smoking habits.[17] Schneiderman and Levin[18] and Burbank and Fraument[13] have suggested "that nonwhites are more exposed or vulnerable than whites to carcinogenic factors" and that one possible etiological factor in relation to other environmental factors to be considered, may be differences in smoking patterns. Existing evidence does not support the smoking hypothesis. The frequency of smokers appears to have been quite similar in 1955 for Northern and Southern blacks and whites.* In fact, only about half as many blacks smoked more than one pack a day as did whites (both in the North and South). The smaller frequency of heavy smokers among blacks is due, most likely, to the small income of blacks (similar findings were reported by Kirchoff[19] and Mills).[19] We would think it reasonable to assume that smoking is completely unrelated to the difference in lung cancer between blacks and whites and migrants and non-migrants.

A second hypothesis is that factors of general malnutrition and related disease may be involved that operate among some migrant, and especially black migrant, populations. Poverty for decades has been the fertile soil for diseases of malnutrition, infectious diseases and associated physiological impairments affecting the susceptibility and adaptability of the individual in subsequent years of life. In this respect, malnutrition in combination with subsequent environmental factors may have some direct bearing on cancer development. This is virtually an unexplored area. Saffiotti et al. found that in experimental animals fed large amounts of vitamin A, lung cancer was inhibited.[20] Conversely, the question arises, would low levels of vitamin A increase the risk

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of lung cancer?

A third possibility exists that virus related disease sustained in childhood or some weak carcinogens which prevailed in the poverty areas during these decades, could be precursors in the subsequent development of cancer after migration, when these individuals are exposed to urban and industrial environments. Experimental as well as clinical evidence exists that the host/virus relationship may be disturbed by external factors. In the consideration of migration, a major question occurs: the possibility that the chemical environment may activate latent tendencies (including perhaps viruses) in humans, which may produce certain diseases as well as cancer.[21-23]

Another hypothesis may be the exposure of migrants to agricultural chemicals during childhood. Southern migrants (especially blacks) largely come from agricultural populations which tend to be heavily exposed to agricultural chemicals. There has been a long and woeful neglect of safety provisions for agricultural laborers, especially relating to their exposure to agricultural chemicals. The relationships between various chemical agents and cancer are too well established for us to discount the possibility that migrant workers may develop lung cancer and other forms of cancer in later years as a response to exposures to agricultural chemicals in the early formative years.

One source of evidence that would speak against this possible major role of agricultural carcinogens comes from the comparison of lung cancer rates in Ohio with those in Southern states. Lung cancer rates for nonwhite are consistently smaller in the South than in Ohio. Similarly, the analysis of the distribution of lung cancer in the U.S. clearly shows that lung cancer rates are much lower in the South Atlantic and Central Southern states than they are in the North Central regions.[24] It is unlikely that these rates would be consistently low if agricultural chemicals were important antecedents of lung cancer, unless the latent period since the introduction of these chemicals has been too short to manifest the carcinogenic effect directly or because of competitive causes of death. Nevertheless, agricultural chemicals may be considered as capable of altering the resistance of the individual to subsequent environmental carcinogenic agents following migration, and requires further study.

In the consideration of the various hypotheses, the industrial environment--exposure to the carcinogenic potential of the chemical environment--chemical dusts, fumes, mists and gases in the work environment, appears as the most compelling and plausible decisive factor in the development of lung cancer among the migrants. Associated with this are factors of urbanization and air pollution.

The considerably higher rate for lung cancer among the males versus the females for those born in Ohio and those born in the South provide strong support to the occupational industrial factor concept. Evidence clearly indicates Southern migrant populations moved into the industrial North for employment. As new recruits with no status, they were given the assignments which invariably exposed them to the adverse stresses of the work environment.

In attempting to reconstruct the life working history among the nonwhites, the term "laborers" as an occupation on death certificates and survey forms, is frequently encountered. It must be recognized that the concept and term of "laborer" as an occupation had different meanings in different industries and geographical areas during three successive decades as a result of the rapid industrialization of the United States.

The term "laborer" as an occupational classification for the nonwhite is misleading in terms of risk and frequently misinterpreted. The complex nature of the working environment within the thousands of diverse, hazardous industries in the North are often not appreciated. Also, not properly understood is the insidious nature of the chemical environment and the delayed biological effects resulting from the dispersion and accumulation of toxic dusts, fumes, mists, gases and wastes throughout various manufacturing processes throughout an entire plant, to which laborers as well as craftsmen and operators are exposed. In this respect, it should be noted that the most rapid rise for the black was the occupation

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category "craftsmen and operators" which was 8% in 1940, 27% in 1950 and 33% in 1960. Within this category, exposure to specific harmful occupational stresses can be more clearly defined.[25] The implications of these aspects on epidemiological studies, warrant the admonition to develop a more appropriate perspective relative to occupational risks among blacks.

We decided to test our hypothesis concerning environmental stresses subsequent to migration against additional data. For this purpose we requested a special analysis of work areas and birth place from the long-term Steel Workers Study (initiated by Lloyd) relative to coke oven workers who had previously been identified as a high risk lung cancer group. The summary statement of the analysis provides strong support for our hypothesis. Among 58,829 men employed at seven Allegheny County steel plants in 1953, there were 2,543 men involved in coke plant operations. Their mortality experience has been followed through 1966. A series of papers has described the methodology of this study and the finding of the excess mortality from lung cancer among coke oven workers.[26]

A strong association exists in this population between birth place and death from lung cancer in the nonwhite coke plant workers. *Examination of these death certificates revealed that 33 of 35 deaths occurred among men born in the South.*[27]

Further support for this hypothesis came from an analysis of the published data of the National Study of Chromate Industry conducted by the Public Health Service.[28]

The number of person years and the number of deaths compared to the expected number for the United States due to respiratory cancer is tabulated.* The ratio of actual to expected number of deaths due to respiratory cancer was fourteen for whites, eighty for nonwhites. The study included data on place of birth which supported the concept of migration for employment for risk occupations particularly for the nonwhite and also for the white. Only five out of 104 black workers in four New Jersey chromate plants were born within that state. One-half of the black employees came from Georgia or South Carolina. Seventeen percent of the Maryland black workers were born in the state and 88% were from North and South Carolina and Virginia.[28]

In our review of lung cancer studies we find that surprisingly little work has been done on the relationship of occupational exposure (and exposure to pollutants) of the thousands of chemicals, individually or in combination, in the chemical environment and subsequent lung cancers during the past three decades. Epidemiologically, there are no studies which encompass the analysis of the total life employment, of the identification and interplay of all the chemicals, dusts and so forth, to which the worker is subjected, in sequential multiple employments in different occupations and manufacturing processes during the life span of an individual.

A recent report states "the number of varieties of worker groups having occupational contact with these carcinogens is considerably larger than indicated by published reports because of the neglect and reluctance of industrial management and most public health agencies to undertake such tasks in the interest of the public good."[29]

Further, the concept has been previously postulated of a combination of two environmental complexes; air pollution and the occupational environment "that air pollution represents a highly probable factor in the excess of lung cancer in urban areas, acting directly and augmenting the occupational exposures of men so that carcinogenic and co-carcinogenic or potentiating agents of both environments may be involved."[30] Compelling evidence along these lines was also presented by Hetticke[31,32] and by Sterling.[33]

It is hoped that the findings from this study will provide the impetus to broaden the concepts and approaches in the consideration of etiological factors associated with lung cancer.

## SUMMARY

Migrants, in particular black migrants, have an increased risk for lung cancer. In a study of Ohio residents, white males born in the South had a 50% excess and nonwhite males 100% excess, compared to those born in Ohio. Yet

*These data can be obtained from the authors.

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there was no difference in the lung cancer rates of black and white Ohio born males.

Implicated are some factors associated with place of birth in the U.S. and in particular in the South and the influences of early years of life and exposure to adverse industrial environments subsequent to migration.

The interaction between environmental and host factors was demonstrated in our analysis of coke oven workers in the prospective steel workers study, which revealed a statistical association with place of birth and high lung cancer among the nonwhite males, 33 of 35 deaths occurred among men born in the South. Further support of migration and risk employment was provided in the analysis of lung cancer among chromate workers.

Our findings support the hypothesis that lung cancer, at least among the blacks, may be largely a result of migration and occupational exposure to chemical dusts and fumes in the industrial employment.

The important evidence to which we now point would indicate that occupational carcinogens may be the immediate antecedents of lung cancer and that region of birth in the U.S., migration and subsequent industrial employment are important variables in the etiology of cancer of the lung.

## ACKNOWLEDGEMENTS

We wish to express our appreciation for the excellent assistance provided by the Ohio Department of Health, in particular, Mr. William E. Voigt, Chief of Division of Vital Statistics and Mrs. Betty Everett, Statistician.

We are also deeply grateful to Dr. Carol Redmond, Graduate School of Public Health, University of Pittsburgh for making available the crucial data on deaths among nonwhite steel workers; to Mr. Arthur LaGasse, University of Pittsburgh, for the computer programming and production and to Dr. Emanuel Landau, Bureau of Radiological Health, Food and Drug Administration, for review of the manuscript.

## LITERATURE CITED

1. BUECHLEY, R. and J.E. DUNN, G. LINDEN, and L. BRESLOW. Excess Lung Cancer Mortality Among Mexican Women in California. Cancer, 10: 63-65, 1957.
2. COX, P. and J.S. GRZYBOWSKI, and B.A. ROWE. Lung Cancer Mortality According to Birthplace. Canad. Assoc. Jour., 99: 476-482, 1955.
3. DEAN, G. Lung Cancer Among White South Africans. Brit. Med. Jour., 2: 852-857, 1959.
4. DEAN, G. Lung Cancer in Australia. Med. Jour. Australia, 1: 1093-1096, 1962.
5. DOLL, R. and D.H. Epidemiology of Lung Cancer in New Zealand. Lancet, 1: 37-39, 1958.
6. HAENSZEL, W. Cancer Mortality Among Foreign Born in the United States. Jour. Nat. Cancer Instit., 26: 37-132, 1962.
7. McCALL, M.G. and W.S. STENHOUSE. Death From Lung Cancer in Australia. Med. Jour. Australia, 10: 524-525, 1971.
8. MANCUSO, T.F. and E.J. COULTER. Cancer Mortality Among Native White, Foreign-Born and Non-White Male Residents of Ohio. Jour. Nat. Cancer Instit., 20: 1958.
9. MANCUSO, T.F. and E.J. COULTER, and E.J. MacDONALD. Migration and Cancer Mortality Experience - A Study of Native and Southern Born Non-White Ohio Residents. In Trace Substances in Environmental Health VI. 1973. A Symposium. D.D. Hemphill, ed., Univ. of Mo., Columbia, 1973.
10. RAKOVER, J. and G. KALLNER. Cancer Mortality and Morbidity in Israel. World Health Organization, Geneva, 1957.
11. SAUERN H. Migration and Risk of Dying. Proceedings Social Statistical Section, Amer. Stat. Ass., 1957, pp. 399-407.
12. HAENSZEL, W. and D.B. LOVELAND, and M.G. SIRKEN. Lung Cancer Mortality as Related to Residence and Smoking Histories, I: White Males. Jour. Nat. Cancer Instit., 28: 947-1001, 1962.
13. BURBANK, F. and J.F. FRAUMENT. U.S. Cancer Mortality: Nonwhite Predominance. Jour. Nat. Cancer Instit. 49: 649-659, 1972.
14. SCHNEIDERMAN, M.A. and D.L. LEVIN. Trends in Lung Cancer, Mortality, Incidence, Diagnosis, Treatment, Smoking and Urbanization. Cancer, 30: 1320-1325, 1972.
15. STERLING, T.D. The Incidence of Lung Cancer in the U.S. Since 1955 in Relation to the Etiology of the Disease. Amer. Jour. Public Health. Feb. 1972.
16. SIEGEL, J.S. Estimates of Coverage of the Population by Sex, Race and Age in the 1970 Census. Demography, 2: Feb. 1974.
17. Cancer Rates in Blacks and Whites. Lancet. (editorial) March 31, 1973, p. 708.
18. KIRCHOFF, H. and R.H. RIGDON. Smoking Habits of 21,642 Individuals in Texas. Jour. Nat. Cancer Institute., 16: 1287-1304, 1956.
19. MILLS, C.A. and M.M. FORTER. Tobacco-Smoking Habits in an American City. Jour. of National Cancer Instit., 13: 1283-1297, 1953.
20. SAFFIOTTI, U. and R. MONTESANO, A. SELLAKUMAR, and S. BORG. Experimental Cancer of the Lung. Cancer, 20: 857, 1967.
21. MANCUSO, T.F. and J.S. MORDELL. Proposed Initial Studies of the Relationships of Community Air Pollution to Health. Environ. Research 2: 102-133, 1969.
22. SPIELGELMAN, S. and R. AXEL, S.C. BONT, R. GULATI, R. HELMAN, D. KEIFF, and J. SCHLON. Human Cancer and the RNA Tumor Viruses. RNA Viruses/Ribosomes. Amsterdam, 1972, pp.73-99.
23. FISHBEIN, L. and W.G. FLAMM, and H.L. FALK. Environmental Effects on Biological Systems. Chemical Mutagens. New York, 1970, p. 364.
24. LILIENFELD, A.M. and J.M.L. LEVIN, and I.I. KESSLER. Cancer in the United States. Vital and Health Statistics Monographs. American Public

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differentiations. The problems faced by the Nigerian patient with untreatable primary amenorrhoea are briefly mentioned.

ACKNOWLEDGEMENT

We are grateful to Professor A.O. Williams and his staff for the histology reports, S.O. Akinkumi of our Paediatric Department for the cytogenetic studies, our medical illustration unit for the photographs, and Miss Cindy Miller of the Johns Hopkins Hospital, Baltimore for the secretarial assistance.

LITERATURE CITED

1. JEFFCOATE, T.N.A. Principles of Gynaecology. Third Ed. Butterworths, London, 1967 pp. 186, 171.
2. McCALL, C.H. and J.M. WAUGH. Absence of the Uterus and Vagina in Association with Left Renal Agenesis. Case Proc. Staff Meeting. Mayo Clinic, 19: 244, 1944.
3. WEI, E.D. Quoted by Coker, O.O. 1966.
4. COKER, O.O. A Case of Congenital Absence of the Uterus. Nigerian Medi Jour., 2: 214-216, 1972.
5. AIMAKHY, V.E. Cytogenetics Studies in Primary Amenorrhoea. Abstracts, 5th International Cytology Congress. Miami, 1974, P. 41.
6. AIMAKHU, V.E. Testicular Feminization Syndrome with and without Clitoral Enlargement. W.A.M.J., (inpress) 1973.
7. JACOBS, P.A. The Chromosome Basis of some Types of Intersexuality in Man. J. Reprod. Fers. Supp., 7: 73-78, 1969.
8. JEFFCOATE, T.N.A. A Clinican's Concept of Intersex Proc. Roy. Soc. Med., 54: 893-897, 1961.
9. JONES, H.W. and W.W. SCOTT. Hermaphroditism, Genital Anomalies and Related Endocrine Disorders. Second Edition, William and Wilkins Co. Baltimore, pp. 193,300, 98.
10. DEWHURST, C.J. and R.R. GORDON. The Intersexual Disorders Bailliere Tindall and Cassell, Lond. 1969 pp. 12,58.
11. HUMPHREY, R.R. Studies on Sex Reversal in Amblystoma: V. The Structure of Ovaries of Amblystoma Tigrinum, Subjected for Long Periods to the Influence of a Testis Resident in the Same Animal. Anat. Rec., 51: 135-145, 1931.
12. HUMPHREY, R.R. Sex Determination in Amblystoma Salamenders: A Study of the Progeny of Females Experimentally Converted into Males. Am. J. Anat., 76: 33-66, 1945.
13. HUMPHREY, R.R. Reversal of Sex in Females of Genotype WW in the Axolotl (Siredon or Amblystoma mexicanum) and its Bearing upon the Role of the Z Chromosomes in the Development of the Testis. J. Exp. Zool., 109: 171-185, 1948.
14. JOST, A. Recherches sur la differenciation Sexuelle de L'embryon de Lapin. I. Introduction et embryologie genitale normale. Arch. Anat. Microsc. Morph. Exp., 36: 151-200, 1947 a.
15. JOST, A. Recherches sur la differenciation Sexuelle de L'embryon de Lapin. II. Action des androgenes de synthese sur L'histogenese genitale. Arch. Anat. Microsc. Morph. Exp., 36: 242-270, 1947 b.
16. JOST, a. Problems of Fetal Endocrinology: The Gonadal and Hypophysical Hormones. Recent. Progr. Horm. Res., 8: 379-418, 1953.
17. JOST, A. Modalities in the Action of Gonadal and Gonadalstimulating Hormones in the Foetus. Mem. Soc. Endocr., 4: 237, 1955.
18. NEWMANN, R. and W. ELGER. Proof of the Activity of Androgenic Agents on the Differentiation of External Genitalia, the Mammary Gland and the Hypothalamic-pituitary System in Rats. Androgens in Normal and Pathological Conditions. Proceedings of the Second Symposium on Steroid Hormones Edited by A.V. Ghent. Publishers Excepta Medical Foundation International Congress Series, 101: 168-185, 1966.
19. NEWMAN, F. and W. ELGER, and H. STEINBECK. Drug Induced Intersexuality in Mammals. J. Reprod. Fert. Suppll., 7: 9-24, 1969.
20. JOST, A. Modalities in the Action of Gonadal and Gonadal stimulating Hormones in the Foetus. Mem. Soc. Endocr., 4: 237, 1955
21. WILLEM, A. VAN N. True Hermaphroditism: Clinical Morphologic and Cytogenetic Aspects. Publisher, Harper & Row, New York, 1974, p. 18.
22. AIMAKHU, V.E. Personal Observations on Eight Cases of True Hermaphroditism, 1974.
23. CARPENTIER, P.J. and E.L. POTTER. Nuclear Sex and Genital Malformations in 48 cases of Renal Agenesis, with Special Reference to Non-specific Female Pseudohermaphroditism. Amer. J. Obstet Gynec., 73: 255-255, 1959.
24. STEWART, D.B. Obstetrics and Gynaecology in the Tropics & Developing Countries. 1st Edition, Edward Arnold, London, 1967, p. 161.
25. LAWSON, J.B. Obstetrics and Gynaecology in the Tropics & Developing Countries. 1st Ed. Edward Arnold, London 1967, p. 547.

(Mancuso & Sterling, from page 111)

Health Association, Harvard Univ. Press, Cambridge, Mass., 1972.
25. PRICE, D.O. Changing Characteristics of the Negro Population. U.S. Bureau of Census, U.S. Government Printing Office, 1960, Census Monograph, Wash., D.C., 1969.
26. LLOYD, J.W. Long Term Mortality Study of Steelworkers V. Respiratory Cancer in Coke Plant Workers. Jour Occupational Med., 13: 53d63, 1971.
27. REDMOND, C. Personal Communication, May 1973.
28. U.S. Public Health Service, Division of Occupational Health: A Study of Health of Workers in chromate Producing Industry No. 192, Wash., D.C., 1953.
29. HUEPER, W.C. in Lawyer's Medical Encyclopedia, Allen Smith, Indianapolis, 1971, p. 560.
30. MANCUSO, T.F. Air Pollution and Cancer. Proceedings of National Conference on Air Pollution, 1958, Public Health Service Pub., No. 648, 1959.
31. HETTCHE, H.O. Schriftreihe der Landes austalt fur Immission, Bodenschutzgebiete des Landes Nordrhein-Westfalen in Essen, 1877, 1970 and 23764, 1971.
32. HETTCHE, H.O. "Luftverunreinigung und Lungenkrebs" Naturvissenshaften, 58: 409-413, 1971.
33. STERLING, T.D. Air Pollution and Smoking. Environment, 15: 3-5, 1973.

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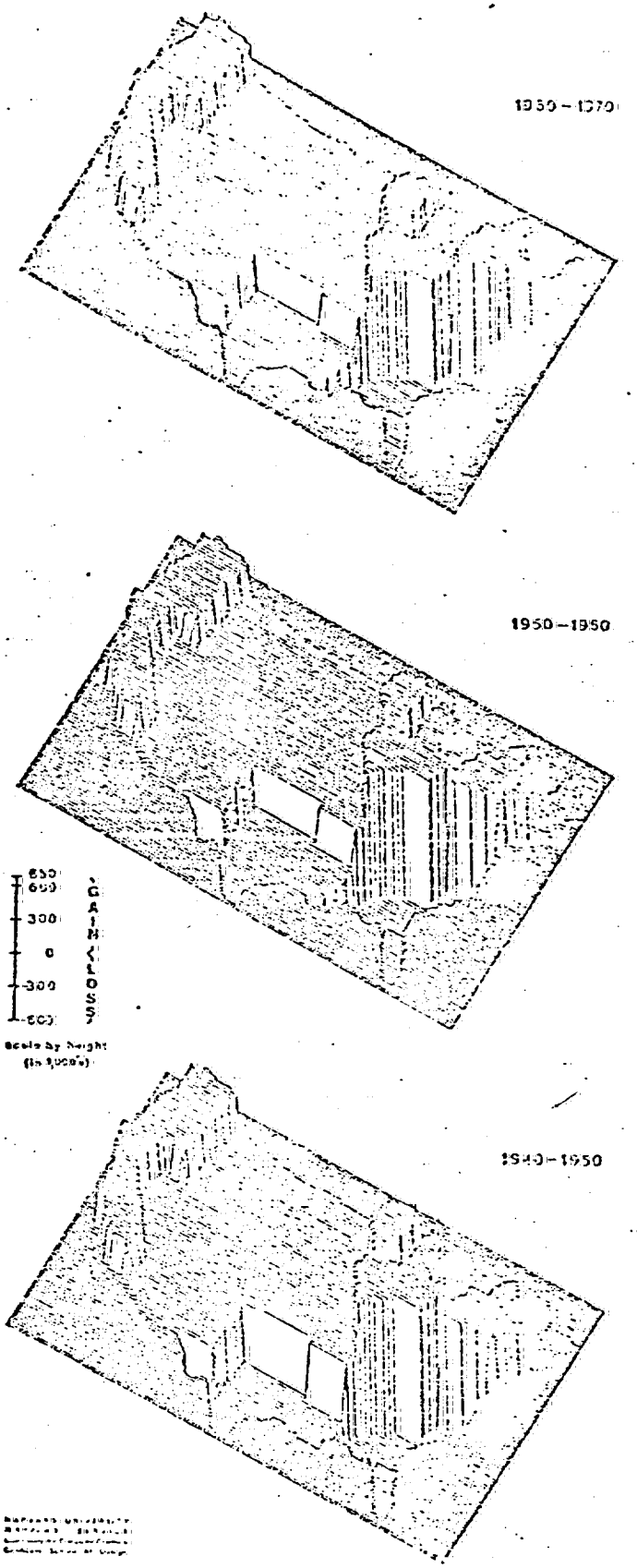


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Table 1

MOVEMENT OF NEGROES, 1940-1970

Net migration among 9 regions (excluding Al[illegible] and Hawaii) as estimated by Bureau of the Census

Table 1

Average Annual Age Specific and Age Adjusted Mortality Rates
for Males from Cancer of Bronchus and Lung (162-163) per 100,000
for Years 1959-1967 for Death of Ohio Residents by Place of Birth and Race

| Age Group | White and Born in Ohio | Nonwhite and Born in Ohio | White and Born in the South | Nonwhite and Born in the South |
|---|---|---|---|---|
| 25 - 29 | 0.43 | 1.11 | 0.47 | 0.88 |
| 30 - 34 | 2.08 | 4.90 | 2.31 | 7.66 |
| 35 - 39 | 6.89 | 15.96 | 10.73 | 15.17 |
| 40 - 44 | 18.04 | 34.70 | 30.29 | 39.80 |
| 45 - 49 | 34.97 | 38.22 | 52.81 | 87.07 |
| 50 - 54 | 78.38 | 55.36 | 113.86 | 161.37 |
| 55 - 59 | 124.19 | 99.14 | 160.58 | 223.73 |
| 60 - 64 | 180.18 | 167.04 | 264.01 | 282.69 |
| 65 - 69 | 221.49 | 213.13 | 290.48 | 413.70 |
| 70 - 74 | 245.46 | 221.69 | 264.25 | 317.45 |
| 75 - 84 | 182.13 | 119.49 | 215.33 | 282.09 |
| 85+ | 111.10 | 40.98 | 127.53 | 114.38 |
| Age Adjusted | | | | |
| 35 - 69 | 77.54 | 73.98 | 108.60 | 145.22 |
| Number of Deaths | | | | |
| 35 - 69 | 6,701 | 157 | 1,924 | 1,137 |
| Age Adjusted | | | | |
| 35+ | 94.66 | 85.36 | 124.95 | 163.95 |
| Number of Deaths | | | | |
| 35+ | 9,210 | 195 | 2,319 | 1,372 |

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Source: https://www.industrydocuments.ucsf.edu/docs/pxmk0000

Table 2

Average Annual Age Specific and Age Adjusted Mortality Rates for Females from Cancer of Bronchus and Lung (162-163) per 100,000 for Years 1959-1967 for Death of Ohio Residents by Place of Birth and Race

| Age Group | White and Born in Ohio | Nonwhite and Born in Ohio | White and Born in the South | Nonwhite and Born in the South |
|---|---|---|---|---|
| 25 - 29 | 0.18 | 0.00 | 0.67 | 0.00 |
| 30 - 34 | 0.87 | 0.97 | 0.89 | 2.03 |
| 35 - 39 | 3.49 | 7.10 | 3.60 | 3.76 |
| 40 - 44 | 5.79 | 12.53 | 11.35 | 9.74 |
| 45 - 49 | 9.51 | 8.07 | 12.99 | 10.38 |
| 50 - 54 | 13.75 | 22.34 | 19.37 | 30.70 |
| 55 - 59 | 15.53 | 16.95 | 31.24 | 21.58 |
| 60 - 64 | 22.67 | 22.05 | 25.40 | 44.22 |
| 65 - 69 | 26.11 | 40.64 | 37.43 | 40.57 |
| 70 - 74 | 31.17 | 21.44 | 43.48 | 34.54 |
| 75 - 84 | 33.80 | 20.44 | 48.07 | 22.61 |
| 85+ | 41.81 | 25.95 | 69.12 | 80.74 |
| Age Adjusted | | | | |
| 35 - 69 | 12.08 | 16.43 | 17.82 | 19.87 |
| Number of Deaths | | | | |
| 35 - 69 | 1,180 | 44 | 344 | 162 |
| Age Adjusted | | | | |
| 35+ | 14.95 | 17.12 | 21.86 | 21.73 |
| Number of Deaths | | | | |
| 35+ | 1,775 | 51 | 454 | 193 |

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Source: https://www.industrydocuments.ucsf.edu/docs/pxmk0000

Table 3

Percent of Cigarette Smokers 18 Years Old and Older by Race and Geographic Region in 1955 [3a]

| | Total | All Cigarette Smokers | Less than One Pack | More than One Pack |
|---|---|---|---|---|
| North and West | | | | |
| White Males | 100 | 45.4 | 32.9 | 12.5 |
| Nonwhite Males | 100 | 48.4 | 40.7 | 7.7 |
| South | | | | |
| White Males | 100 | 48.4 | 35.7 | 12.7 |
| Nonwhite Males | 100 | 43.1 | 37.8 | 5.3 |
| North and West | | | | |
| White Females | 100 | 24.9 | 22.3 | 2.6 |
| Nonwhite Females | 100 | 30.8 | 28.0 | 2.8 |
| South | | | | |
| White Females | 100 | 19.6 | 17.7 | 1.9 |
| Nonwhite Females | 100 | 16.6 | 15.9 | .7 |

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Source: https://www.industrydocuments.ucsf.edu/docs/pxmk0000

Table 4

Average Age Adjusted Mortality (per 100,000 of Population, all ages) for Cancer of the Bronchus and Lung (162-163) by sex, race for Ohio and Southern States for Years 1950-1967 [35]

| State | MALE | | FEMALE | |
|---|---|---|---|---|
| | White | Non-White | White | Non-White |
| Ohio | 16.04 | 24.34 | 2.28 | 2.96 |
| Alabama | 22.31 | 14.63 | 3.14 | 2.15 |
| Florida | 17.94 | 14.43 | 2.30 | 1.48 |
| Georgia | 20.20 | 12.79 | 2.78 | 1.82 |
| Louisiana | 19.29 | 14.38 | 1.78 | 1.51 |
| Mississippi | 14.93 | 6.94 | 1.81 | 1.12 |
| North Carolina | 14.88 | 9.77 | 2.13 | 1.90 |
| South Carolina | 13.13 | 7.12 | 1.63 | 0.95 |
| Tennessee | 14.46 | 13.21 | 1.63 | 1.77 |
| Texas | 20.35 | 16.29 | 2.96 | 2.39 |
| Virginia | 13.29 | 10.39 | 1.89 | 1.32 |
| United States | 16.28 | 16.30 | 2.22 | 2.23 |

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Source: https://www.industrydocuments.ucsf.edu/docs/pxmk0000

Table 5

Observed and Expected Number of Deaths and Relative Risk of Dying from Lung Cancer, 1953-1966, Among Men Employed in the Coke Plant in 1953 in Allegheny County, Pennsylvania.

| Birthplace | No. at Risk | Observed Deaths | Expected Deaths | Relative Risk |
|---|---|---|---|---|
| | | WHITE | | |
| Pennsylvania | 1311 | 6 | 8.5 | 0.70 |
| Other U.S. | 163 | 2 | 1.5 | 1.32 |
| Foreign Born | 158 | 1 | 3.1 | 0.32 |
| | | NONWHITE | | |
| Pennsylvania | 182 | 0 | 1.9 | -- |
| Other U.S. | 721 | 35 | 10.1 | 4.25* |
| Foreign Born | 2 | 0 | -- | -- |

* Summary Chi-Square Significant at the 1% level.

Expected numbers of deaths are based on the age-race specific rates among the total steelworkers cohort.

There were six coke plant workers with unknown birthplaces.

(Data by personal communication from Dr. Carol Redmond from a study supported in part by Contract No. HSM 099-71-32)

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Source: https://www.industrydocuments.ucsf.edu/docs/pxmk0000

Table 6

Number of deaths from respiratory cancer except larynx, by color and age, among male members of sick benefit organizations in six chromate-producing plants compared with the expected number based on the average death rate for the United States, 1940-1948, inclusive[1] for ages 15-74 years.

[The chromate data include experiences of two plants during 1940-1948, one plant during 1943-1948, three plants during 1946-1948].

| | Number of Deaths | | Ratio of actual to expected number | Annual Number of Deaths per 100,000 males | |
|---|---|---|---|---|---|
| | Actual | Expected[2] | | Chromate | U.S. |
| All Males | 26 | 9 | 28.89 | 470.8 | 16.7 |
| White Males | 10 | 7 | 14.29 | 261.6 | 17.4 |
| Black Males | 16 | 2 | 80.00 | 941.7 | 10.0 |

[1] Violent and accidental deaths not included.

[2] The average death rate for the United States for the 9 years, 1940-1948, multiplied by the appropriate person-years of membership in the chromate plants.

Person-Years of Membership in Sick Benefit Association of Chromate Workers by Color and Age

| Color | Total 15-74 | Age Group (years) 15-34 | 35-44 | 45-54 | 55-64 | 65-74 |
|---|---|---|---|---|---|---|
| | Average number of person-years of membership | | | | | |
| All Males | 5,522 | 1,574 | 1,616 | 1,411 | 796 | 125 |
| White Males | 3,823 | 1,041 | 1,091 | 919 | 659 | 113 |
| Black Males | 1,699 | 533 | 525 | 492 | 137 | 12 |

Abstracted from U.S. Public Health Service, Division of Occupational Health: A Study of Health of Workers in Chromate Producing Industry.[33]

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Source: https://www.industrydocuments.ucsf.edu/docs/pxmk0000